

Benign Conduction Abnormalities in Response to Acute, Moderately-High, Simulated Altitude Exposure

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ABSTRACT

Acclimatization to altitude can improve endurance performance above levels achieved solely by training at sea level. There is natural limitation in the applicability of employing terrestrial altitude training – namely proximity. A simple, non-cumbersome method of simulating altitude is desirable to many types of endurance athletes. The Alto2Lab (Pharma Pacific Inc.), consisting of primarily a breathing tube and silo stack, has shown some potential in this role. There is a lack of evidence regarding whether simulated altitude exposure triggers abnormal cardiovascular responses. The aim of this study was to provide initial evidence of cardiac changes associated with usage patterns that follow distributor guidelines. Twenty-five participants (mean age 29 ± 10.7 ; 16 males; 9 females) volunteered for the study. Subjects underwent a baseline ECG recording followed by ECG recording during sham (4-5 mins), hypoxia (~6 mins), and recovery (3-4 mins) phases. The sham phase consisted of subjects breathing normoxic air through a foam-filled silo system. The sham stack mimicked the look and feel of the silo system used to produce hypoxia with the difference being a single, soda lime-filled silo. A recovery phase followed hypoxia. Pulse oximetry (SpO₂) was used to assess oxygen saturation. Cochran's Q was employed to test the frequencies of responses across the phases. An independent, blinded, experienced clinician (DK) analyzed the recordings. Two subjects were removed from the final analysis (inability to finish the protocol, baseline right bundle branch block). All subjects demonstrated an increase in heart rate (mean = $+16.8 \pm 8.0$) during the hypoxia (mean oxygen saturation = $82 \pm 4.1\%$) phase. No ECG ischemic changes were seen across any of the phases. Benign conduction abnormalities (sinus arrhythmia = 9; junctional rhythms = 4) occurred with some regularity during hypoxia. These abnormalities occurred with less frequency during the sham and recovery phases. It is possible that an altered breathing pattern or an inadequate washout period between phases might account for these findings. Overall, there was no significant relationship between the heart response and phase ($p = .375$). While the Alto2Lab did not produce any ECG changes indicative of an ischemic response, the present study used a small sample of healthy, recreationally-active participants. A larger study employing patients among higher risk categories would provide data that is not currently present in the literature and to which this trial cannot speak.